156 **Editorials**

- Gerbase AC, Rowley JT, Mertens TE. Global epidemiology of sexually transmitted diseases. *Lancet* 1998;351 (suppl III):2–4.
 Brunham RC, Paavonen J, Stevens CE, et al. Mucopurulent cervicitis—the ignored counterpart in women of urethritis in men. N Engl J Med 1984;311:1–6.
- 3 Mårdh PA, Ripa KT, Svensson L, et al. C trachomatis infection in patients with acute salpingitis. N Engl J Med 1977;296:1377-9.
 4 Paavonen J, Lehtinen M. Chlamydial pelvic inflammatory disease. Human
- Reprod 1996;2:519-29.

 5 Cates W, Wasserheit JN. Genital chlamydial infections: epidemiology and
- reproductive sequelae Am J Obstet Gynecol 1991;164:1771–81.
 6 Brinton LA. Epidemiology of cervical cancer—overview. IARC Sci Publ 1992;
- 119:3-23.
- 7 Walboomers JMM, Jacobs MV, Manos MM, et al. Human papillomavirus is a necessary cause of invasive cervical cancer worldwide. FPathol 1999;189:
- 8 Wallin K-L, Wiklund F, Ångstöm T, et al. Type-specific persistence of human papillomavirus DNA before the development of invasive cervical cancer. N Engl J Med 1999;341:1633–8.

- cancer. N Engl J Med 1999;341:1633–8.
 9 Josefsson AM, Magnusson PKE, Ylitalo N, et al. Viral load of human papilloma virus 16 as a determinant for development of cervical carcinoma in situ: a nested case-control study. Lancet 2000;355:2189–93.
 10 Paavonen J, Vesterinen E, Mayer B, et al. Genital Chlamydia trachomatis infection in patients with cervical atypia. Obstet Gynecol 1979;54:289–71.
 11 Schachter J, Hill EC, King EB, et al. Chlamydia trachomatis and cervical neoplasia. JAMA 1982;248:2134–8.
 12 Kiviat N, Paavonen J, Brockway J, et al. Cytologic manifestations of vaginal and cervical infections. Epithelial and inflammatory cellular changes. JAMA 1985;253:989.
 13 Hakama M, Lehting M, Knekt P, et al. Serum antibodies and subsequent.
- JAWA 1985;253:969.
 Jakama M, Lehtinen M, Knekt P, et al. Serum antibodies and subsequent cervical neoplasms. A prospective study with 12 years follow-up. Am J Epidemiol 1933;137:166–70.
 Lehtinen M, Dillner J, Luostarinen T, et al. Nested case control study of the role of human papillomavirus type 16 and Chlamydia trachomatis in cervical carcinoma. BMJ 1996;312:537–9.
- 15 Koskela P, Anttila T, Bjørge T, et al. Chlamydia trachomatis infection and invasive cervical carcinoma. Int J Cancer 2000;85:35–9.
 16 Anttila T, Saikku P, Koskela P, et al. Serotypes of Chlamydia trachomatis and the risk for the development of cervical squamous cell carcinoma. JAMA 2001;285:47–51.
 17 Risch HA, Marrett LD, Howe GR. Parity, contraception, infertility, and risk
- 17 National A. J. Howe of a Tarthy, contaception, intertuny, and risk of epithelial ovarian cancer. Am J Epidemiol 1994;140:585–97.
 18 Paavonen J, Kiviat N, Wölner-Hanssen P, et al. Colposcopic manifestations of cervical and vaginal infections. Obstet Gynecol Surv 1988;43:373–81.
 19 Bjorge T, Dillner J, Anttila T, et al. A prospective seroepidemiological study
- of the role of human papillomavirus in non-cervical anogenital cancers. BMJ 1997;315:646–9.
- 20 Rossing MA, Daling JR, Weiss NS. Ovarian tumors in a cohort of infertile women. N Engl J Med 1994;331:771–806.
 21 Whittemore AS. The risk of ovarian cancer after treatment for infertility. N
- Engl J Med 1994;331:805-6.

- 22 Hankinsson SE, Hunter DJ, Colditz GA, et al. Tubal ligation, hysterectomy, and risk of ovarian cancer. JAMA 1993;270:2813–18.
 23 Whittemore AS, Harris R, Itnyre J, and the Collaborative Ovarian Cancer Group. Characteristics relating to ovarian cancer risk: collaborative analysis of 12 US case-control studies. Am J Epidemiol 1992;136:1184–203.
 24 Franceschi S, La Vecchia C, Negri E, et al. Fertility drugs and risk of epithelial ovarian cancer in Italy. Hum Reprod 1994;9:1673–5.
 25 Mosgaard BJ, Lidegaard Ø, Kruger Kjaer S, et al. Infertility, fertility drugs, and invasive ovarian cancer: a case-control study. Fertil Steril 1997;67: 1005–12.

- Modan B, Ron E, Lerner-Geva L, et al. Cancer incident in a cohort of infertile women. Am J Epidemiol 1998;147:1038–42.
 Venn A, Watson L, Bruinsma F, et al. Risk of cancer after use of fertility drugs with in-vitro fertilisation. Lancet 1999;354:1586–90

- drugs with in-vitro fertilisation. Lancet 1999;354:1586–90
 28 Bosch FX, Cardis E. Cancer incidence correlations: genital, urinary and some tobacco-related cancers. Int J Cancer 1990;46:178–84.
 29 Paavonen J, Miettinen A, Heinonen PK, et al. Serum CA 125 in acute pelvic inflammatory disease. Br J Obstet Gynecol 1989;96:574–9.
 30 Paavonen J, Lehtinen M, Lehto M, et al. Concentrations of tumorassociated trypsin inhibitor and C-reactive protein in serum in acute pelvic inflammatory disease. Clin Chem 1989;35:869–71.
 31 Risch HA, Howe GR. Pelvic inflammatory disease and the risk of epithelial ovarian cancer. Cancer Epidemiol Biomark Prev 1995;4:447–51.
 32 Parazzini F, La Vecchia C, Negri E, et al. Pelvic inflammatory disease and risk of ovarian cancer. Cancer Epidemiol Biomark Prev 1996;5:667–9.
 33 Rosin MP, Anwar WA, Ward JA. Inflammation, chromosomal instability and cancer: the schistosomiasis model. Cancer Res 1994;54 (Suppl):1929–33.
 34 Schachter J. LGV and cancer. In: Schachter J, Dawson CR, eds. Human chlamydial infections. Littleton: PSG Publishing Co, 1978:56–7.
 35 Eck M, Schmausser B, Haas R, et al. MALT-type lymphoma of the stomach

- 35 Eck M, Schmausser B, Haas R, et al. MALT-type lymphoma of the stomach is associated with Helicobacter pylori strains expressing the CagA protein. Gastroenterology 1997;122:1482-6.
 36 Chow WH, Blaser M, Blot W, et al. An inverse relation between cagA strains
- of Helicobacter *pylori* infection and risk of esophageal and gastric cardia adenocarcinoma. *Cancer Res* 1998;**58**:588–90.
- 37 Paavonen J, Lehtinen M. Immunopathogenesis of chlamydial pelvic inflammatory disease. The role of heat-shock proteins. Infect Dis Obstet Gynecol 1994;2:105–10. 38 Lan J, Melgers CJ, Walboomers JM, et al. Prevalence and serovar
- distribution of asymptomatic cervical Chlamydia trachomatis infections as determinant by highly sensitive PCR. J Clin Microbiol 1995;33:3194–7.
- Dean D, Oudens E, Bolan G, et al. Major outer membrane protein variants of Chlamydia trachomatis are associated with severe upper genital tract infection and histopathology in San Francisco. J Infect Dis 1995;172:1013–
- 40 Mayer JM, Woods ML, Vavrin Z, et al. Gamma interferon-induced nitric oxide production reduces Chlamydia trachomatis infectivity in Mc Coy cells. Infect Immun 1993;61:491–7.
- 41 Fan B, Lu H, Hu H, et al. Inhibiton of apoptosis in Chlamydia-infected cells: blockade of mitochondrial cytochrome c release and caspase activation. \mathcal{J} Exp Med 1998;187:487-96.

Sector-wide approaches and STI control in Africa

Allocation of aid from international agencies to the health sector in developing countries has usually involved funding of specific projects. This process enabled donors to suggest priorities and to monitor accountability of spending. More recently, a different system using sector-wide approaches (SWAps) has been adopted by an increasing number of funders including the World Bank, World Health Organization, and the Department for International Development. Through SWAps, funds are given to the entire health sector for priorities determined by ministries of health rather than to specific projects.1 In theory the system should lead to greater efficiency through reduction of duplicative mechanisms that may occur through multiagency support.

Most of the UN agencies now recognise HIV increasingly as a societal problem. This belief would therefore seem to justify the allocation of HIV prevention funds to the whole health sector across the board. SWAps also appear justified by the contention that HIV/AIDS is associated with poverty and that the poor are more likely to access services that can be delivered at the primary healthcare level. Furthermore, this approach offers all HIV interested parties or stakeholders an opportunity to obtain funds from a central pool and have an input into HIV prevention strategies.

Serious doubts remain, however, about whether SWAps are effective.2 No evaluation of SWAps in STI control has been undertaken. While the role of STI in preventing HIV is now well established, there are still conflicting opinions and uncertainty about how STI services for the population are best delivered. Clear policy directives are even more difficult to justify following the contrasting results of the Mwanza and Rakai studies in which both STI control strategies and the relative effects of the interventions differed significantly.3 4 Given these uncertainties, will SWAps be a good idea for improving STI control in developing countries, and, more importantly, those communities with significant STI/HIV problems?

To answer this one must firstly look at the wider public health aspects of STI control and acknowledge the diversity of the HIV and STI epidemics. In Africa the prevalence of STIs appears to vary significantly between countries and populations. The prevalence of genital ulcer disease is higher in the countries worst affected by HIV in Africa.⁵ Clearly, in some countries STI are a major problem and require a special focus while in others they are of lesser importance. In countries with significant STI/HIV epidemics, some of the potential concerns in adopting SWAps are as follows.

Lack of advocacy

In Africa there are few specialist physicians in STI/HIV. Historically, the majority of African countries have accorded little importance to STI in health budgets. This may reflect a state of denial and a belief that because STIs are not life threatening, individuals who brought such problems upon themselves did not merit special treatment and deserved to be punished for their immoral actions. Such notions are well established in many communities **Editorials** 157

and may be resistant to change, despite the recent evidence supporting STI interventions against HIV. If the project based approach is to be abandoned, ring fencing of funds for STI control activities may well be required.

The importance of advocacy is well demonstrated by the current situation in South Africa where local policymakers have adopted a low key approach to STI/ HIV/AIDS. HIV is still not accepted as a major problem despite antenatal prevalences of more than 30% in some provinces. This denial at the highest governmental level is causing considerable distress and confusion among professionals working in the HIV field and the population at large.

Dilution of expertise

Responsibility for STI control may rest with a number of agencies including AIDS/HIV prevention, clinical services and reproductive health (RH), and others. The expanded concept of joint services for STI and RH has received strong political support, not least because they are directed mainly at women who are perceived to be victims of the HIV epidemic. However, in assessing whether integration is the way forward, limitations as well as benefits should be acknowledged.7 While recognising that RH clinics may provide expertise designed for primary health care, they have little experience of providing specialised services for men. It is also worrying that the limited numbers of medical posts in STI have already been reduced in some areas following decentralisation of services.8

Targeting HIV core groups will be curtailed

Traditionally targeting has focused on groups such as sex workers and their clients, truck drivers and the military. As the epidemic expands out to the general population, new core groups at high risk of HIV must be identified in the community. One such group is STI clinic attenders—in some areas there is evidence that 77% of men with genital ulcers9 and more than 60% of routine attenders are HIV positive. 10 STI services for men, a crucial group for targeting in urban settings, need dramatic improvement. 11 Designated clinics are also justified for a number of reasons, including enhanced surveillance, antibiotic susceptibility testing, training and education, referral of problem cases, evaluation of syndromic management protocols, and as a centre to develop expertise for an STI control programme.

A limited number of multisectoral STI interventions—for example, the Mwanza project, have been implemented usually through donor funding. The Mwanza intervention involved vertical and horizontal programmes but was undertaken in a rural population with a relatively low HIV prevalence for sub-Saharan Africa (4%). Whether the favourable results seen in this study would be replicated elsewhere in countries with worse HIV epidemics is unknown.

Providing scope for targeted interventions is a crucial component of a successful strategy for many countries.¹² However, SWAps are likely to limit the acceptance of targeting which is recognised as among the most cost effective strategies for STI/ HIV prevention. In Thailand, new STI clinics were opened and contributed to the success of the 100% condom use campaign. While the dynamics of the spread of HIV in Thailand are different in Africa, there is a strong case to be made for increasing the number of STI clinics in urban centres in the latter. In South Africa, targeted programmes for sex workers and miners brought about significant reductions in STI incidence.¹³ Other established services for vulnerable and possibly illegal core groups such as sex workers, street kids, and injecting drug users could also suffer through SWAps. Again, ring fencing of funds by policymakers would appear to be necessary to assist these groups through STI and HIV prevention projects.

Supply and distribution of STI drugs

The importance of STI drugs in supporting a programme should not be underestimated. Drug shortages can quickly lead to both a loss of credibility of a programme and falling morale among service providers. Also, the potential for slippage (theft) of drugs may be considerable. Demand for STI drugs may be almost inexhaustible if used prophylactically. STI drugs provided for distribution to the whole health sector would be very difficult to track. Effective monitoring of drug use is paramount and should also be capable of standing up to rigid assessment through independent evaluation.

Conclusion

We are still not clear what is the best approach to improve STI control. The evidence base and what constitutes good governance in STI control varies significantly between populations. While SWAps support the horizontal approach to STI control by strengthening primary healthcare services, sometimes at the expense of designated specialist services, there is no evidence that this is the best strategy. Perhaps STI programme planners should take note of how health sector reform may affect tuberculosis control. Decentralisation of tuberculosis services into primary health care in Zambia has led to a marked reduction in funding and a deterioration in services for people with tuberculosis.14 Optimal use of scarce resources for STI control probably requires a combination of approaches involving aspects of both the horizontal and vertical systems taking into account their strengths and weaknesses.15 16 The tried and trusted methods in reducing STIs that have been poorly implemented in many African countries with severe STI/HIV problems should not be ignored in deference to SWAps until a full evaluation of its effectiveness has been undertaken.

NIGEL O'FARRELL

Pasteur Suite, Ealing Hospital, Uxbridge Road, London UB1 3HW, UK ofarrell@postmaster.co.uk

1 Cassels A, Janovsky K. Better health in developing countries: are sector-wide approaches the way of the future? Lancet 1998;352:1777-9.

2 Garner P, Flores W, Tang S. Sector wide approaches in developing countries. BMJ 2000;321:129-30.

- 3 Grosskurth H, Mosha F, Todd J, et al. Impact of improved treatment of sexually transmitted diseases on HIV infection in rural Tanzania: randomized controlled trial. Lancet 1995;346:530–6.
- Wawer MJ, Sewankambo NK, Serwadda D, et al. Control of sexually transmitted diseases for AIDS prevention in Uganda: a randomised community trial. *Lancet* 1999;353:525–36.

 5 O'Farrell N. The need for targeted interventions against genital ulcers in
- countries worst affected by the HIV epidemic in Africa. Bull World Health Organ (in press).

 6 Editorial. Politicisation of debate on HIV care in South Africa. Lancet 2000;

355:1473

7 MacNeil J. Integrating STD services into reproductive health services. In: Dallabetta G, Laga M, Lamptey P, eds. Control of sexually transmitted diseases. A handbook for the design and management of programs. AIDSCAP/Family Health International, 1996. O'Farrell N, Hoosen AA. Sexually transmitted diseases in South Africa: epidemic donovanosis in Durban. Genitourin Med 1997;73:76.

9 Latif AS. A report on a study to determine the aetiology and pattern of STD amongst men and women presenting to health centers in Harare, Zimbabwe, and to determine risk factors for cervicitis among symptomatic and asymptomatic women. Unpublished report 1994.

10 Karita E, Van de Perre P, Nziyumvira A, et al. HIV prevalence among STD patients in Kigali, Rwanda during the 4-year period 1988–1991. Eighth International Conference on AIDS, Amsterdam, Abstract PoC 4468, 1992.

11 O'Farrell N. Enhanced efficiency of female to male HIV transmission in

- core groups in developing countries: the need to target men. Sex Transm Dis 2001;28:84-91 12 Sumartojo E, Carey JW, Doll LS, et al. Targeted and general population interventions for HIV prevention: towards a comprehensive approach.
- AIDS 1997;11:1201–9.

 Steen R, Vuylsteke B, DeCoito A, et al. Evidence of declining STD prevalence in a South African mining community following a core group intervention. Sex Transm Dis 2000;27:1-8.

14 Bosman MC. Health sector reform and tuberculosis control: the case of Zambia. Int J Tuberc Lung Dis 2000;4:606–14.
 15 Van Praag E, Knippenberg R. Issues in integration of STD programs into health-care systems. In: Dallabetta G, Laga M, Lamptey P, eds. Control of sexually transmitted diseases. A handbook for the design and management of programs. AIDSCAP/Family Health International, 1996.

16 WHO/UNAIDS. Sexually transmitted diseases: policies and principles for prevention and care. Best practice collection. WHO/ UNAIDS, 1997.